American Journal of Medical Case Reports, 2021, Vol. 9, No. 4, 253-258
Available online at http://pubs.sciepub.com/ajmcr/9/4/12
Published by Science and Education Publishing
DOI:10.12691/ajmcr-9-4-12

Chest Pain Radiating to the Jaw with Elevated Troponin: Aortic Dissection Masquerading as Myocardial Infarction

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Received January 03, 2021; Revised January 27, 2021; Accepted February 04, 2021

Abstract Introduction: Chest pain radiating to the jaw, is a classic symptom of acute myocardial infarction (AMI). Electrocardiogram (EKG) changes consistent with ischemia and elevated serum troponin I levels are used to confirm the diagnosis. Prompt management including medical or procedural intervention is required to help reduce mortality in patients with AMI. However, additional conditions may mimic those of classic primary coronary pathology. Case Report: We present the case of a 43-year-old male who came to the emergency department (ED) complaining of acute onset of chest pain radiating to the left jaw. His EKG revealed mild ST changes and T-wave inversion in the antero-lateral leads. Laboratory testing revealed an elevated serum troponin level of 0.18 ng/mL (normal: < 0.04 ng/mL). He was taken for a coronary angiogram and during the procedure, an aortogram was performed revealing a type A aortic dissection. Findings were confirmed by Computed Tomography Angiogram (CTA) showing a dissection extending from the aortic root to the iliac bifurcation. He was promptly taken to the operating room (OR) for surgical repair. Conclusion: Aortic dissection (AD) is included in the differential diagnosis in patients presenting with symptoms and signs consistent with AMI. However, in patients presenting with AD, there are some clues in history and physical exam, such as high blood pressure, history of noncompliance with antihypertensive medications, or location of pain that may provide a clue towards an underlying AD. Our case demonstrates that even if all evidence points towards an AMI, AD should still be on the differential until successfully ruled out by imaging.

Keywords: *Aortic Dissection, Myocardial Infarction, Point-of-care ultrasound, POCUS*

Cite This Article: Sufyan AbdulMujeeb, Faisal Masood, Dureshahwar Ali, Sarah Doleeb, and Adib Chaus, "Chest Pain Radiating to the Jaw with Elevated Troponin: Aortic Dissection Masquerading as Myocardial Infarction." *American Journal of Medical Case Reports*, vol. 9, no. 4 (2021): 253-258. doi: 10.12691/ajmcr-9-4-12.

1. Case Report

A 43-year-old male with a past medical history of hypertension (not on any medication) presented to the ED complaining of substernal chest pain that started approximately three hours prior to presentation. He described the pain as sudden onset and radiating to the left jaw. He rated the pain on a scale of 1-10 as 8/10, with 10 being the worst. He had no other complaints. His blood pressure upon arrival was 144/52. Laboratory data revealed an elevated troponin of 0.18 ng/mL. His EKG revealed mild ST changes in the anterolateral leads along with T-wave inversions (Figure 1). His family history was significant for AMI in his father that required 5 stents. Chest x-ray (CXR) showed mild cardiomegaly, but no widened mediastinum (Figure 2). Given the strong family history of AMI and the classic AMI presentation of chest pain radiating to the jaw, it appeared that the patient was having an acute coronary syndrome, but not necessarily a true ST-Elevation MI (STEMI). Prompt intervention was indicated and he was started on a titratable nitroglycerin drip and a heparin infusion with a bolus. The initial goal was to titrate the chest pain to 0/10 with the nitroglycerin drip while getting frequent EKGs to assess for the development of a true STEMI. Multiple repeated EKGs continued to show findings similar to the initial EKG. After about 5-6 hours of ongoing chest pain despite uptitrating the nitroglycerin drip, it was decided to perform a coronary angiogram.

During the procedure, engagement of the right and left coronaries was extremely difficult. Therefore, an aortogram was performed. This showed evidence of dilated aortic root, aortic insufficiency, and an intimal flap suggesting possible ascending AD (Figure 3). The procedure was terminated and an emergent CTA was performed to rule out AD. CTA showed a type A dissection of the aorta extending from the aortic valve to the bifurcation of both common iliac arteries (Figure 4 & Figure 5). Additionally, the dissection also involved the left subclavian and the right innominate artery (Figure 6). The patient was immediately taken to the OR for surgical repair of this massive dissection. Post operatively, the

patient recovered well and he was discharged home without any significant complications.

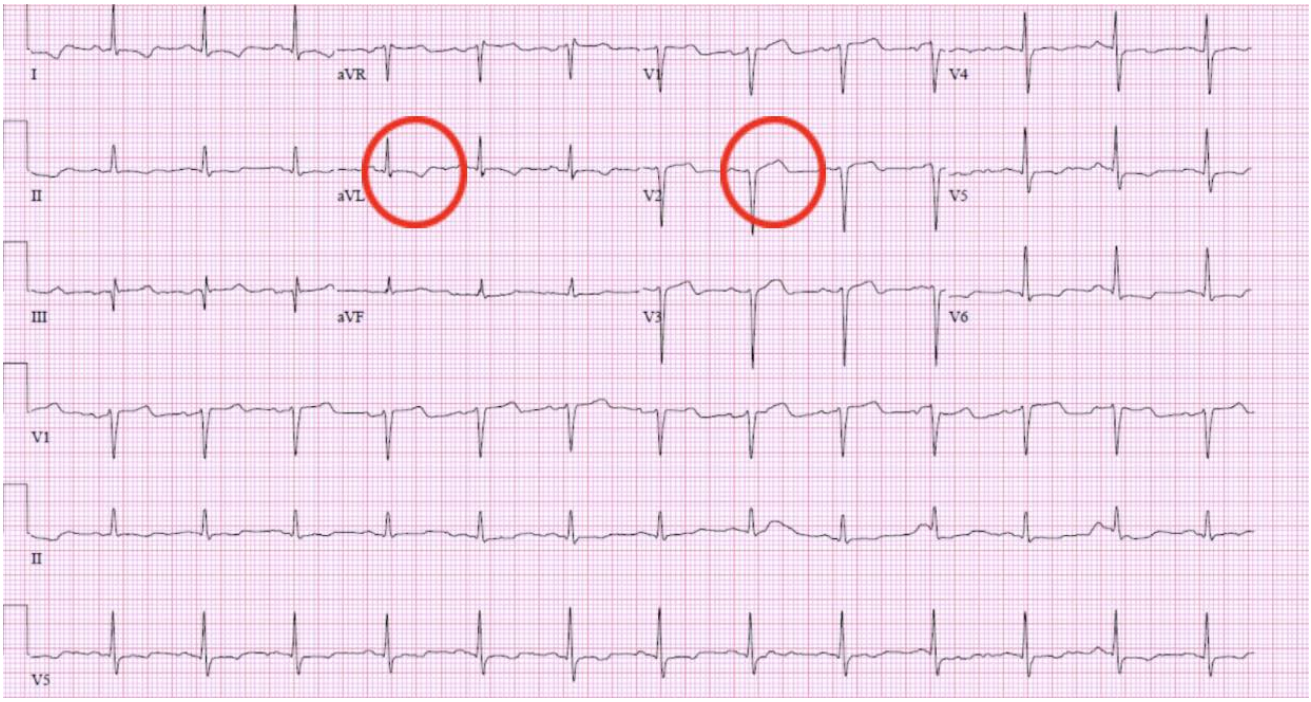

Figure 1. EKG with mild ST changes and T-wave inversion in anterolateral leads

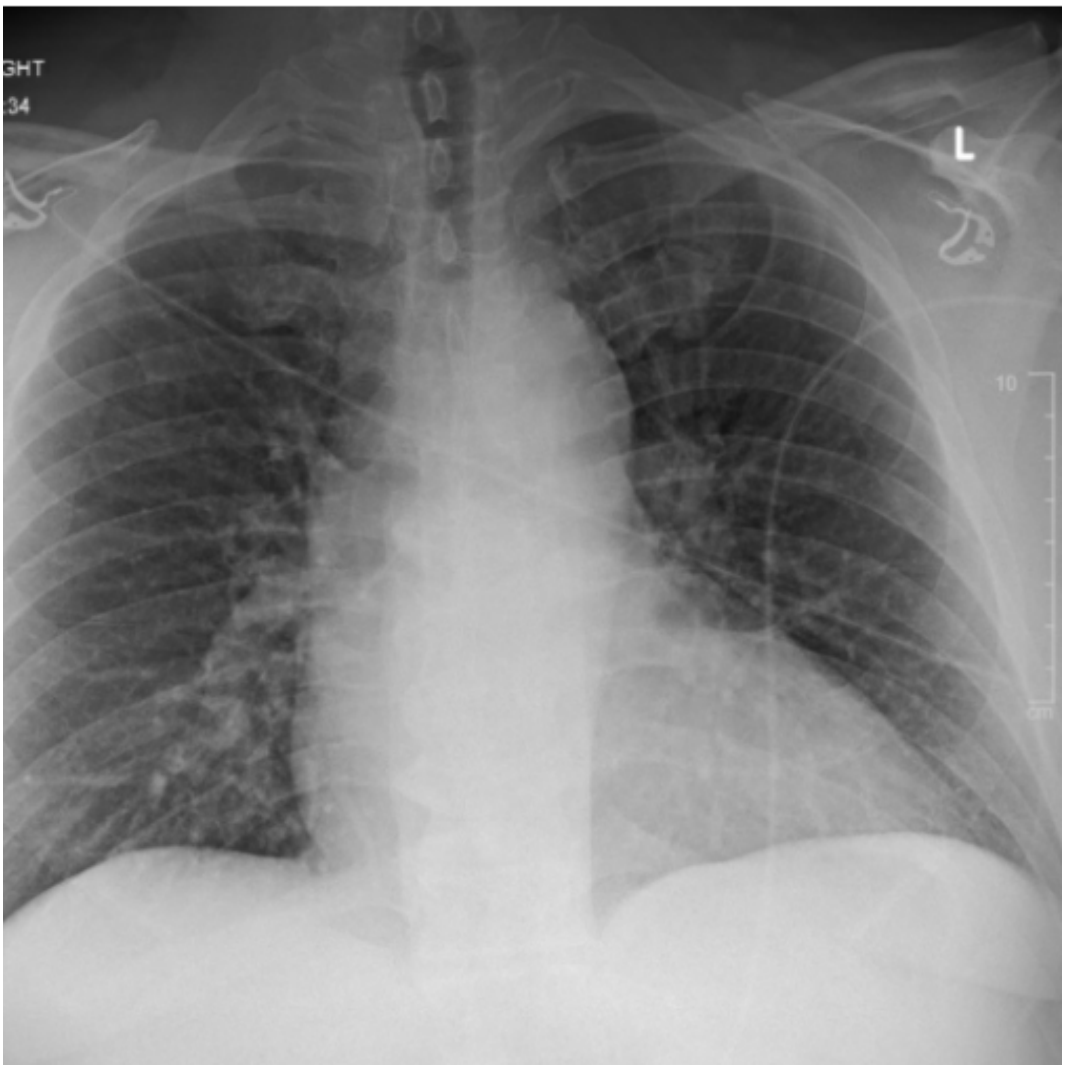

Figure 2. CXR with mild cardiomegaly and no widened mediastinum

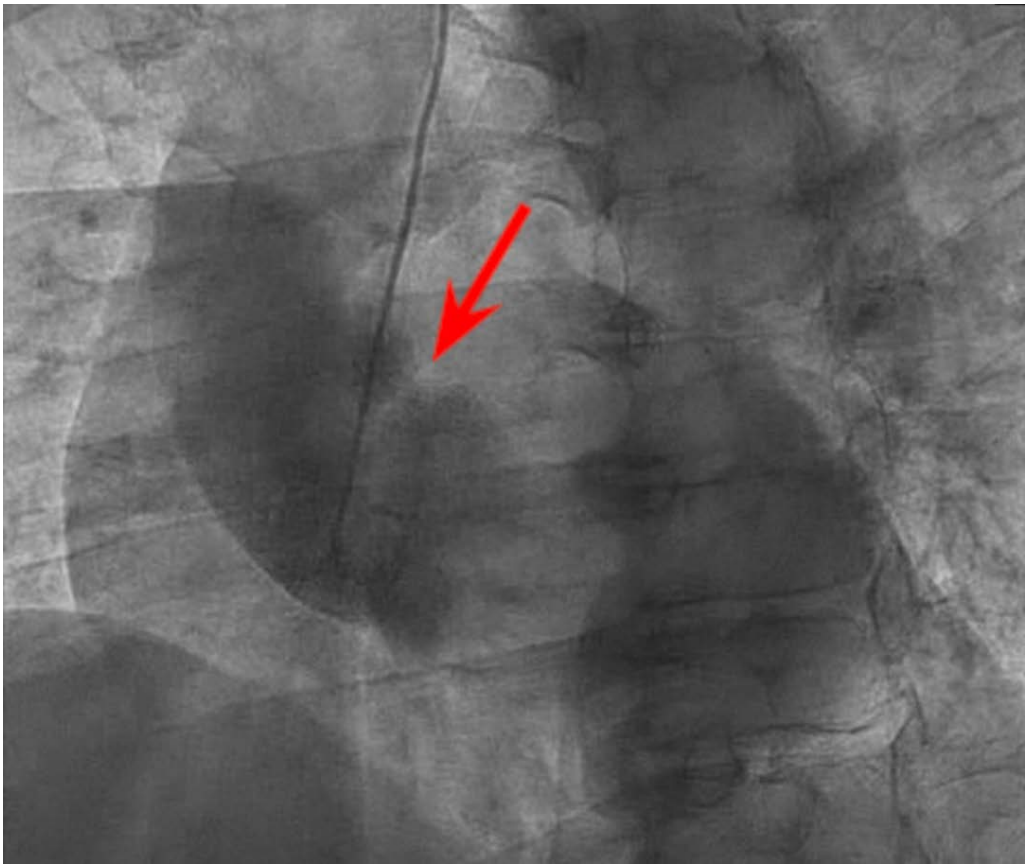

Figure 3. Aortogram performed during catheterization showing an intimal flap

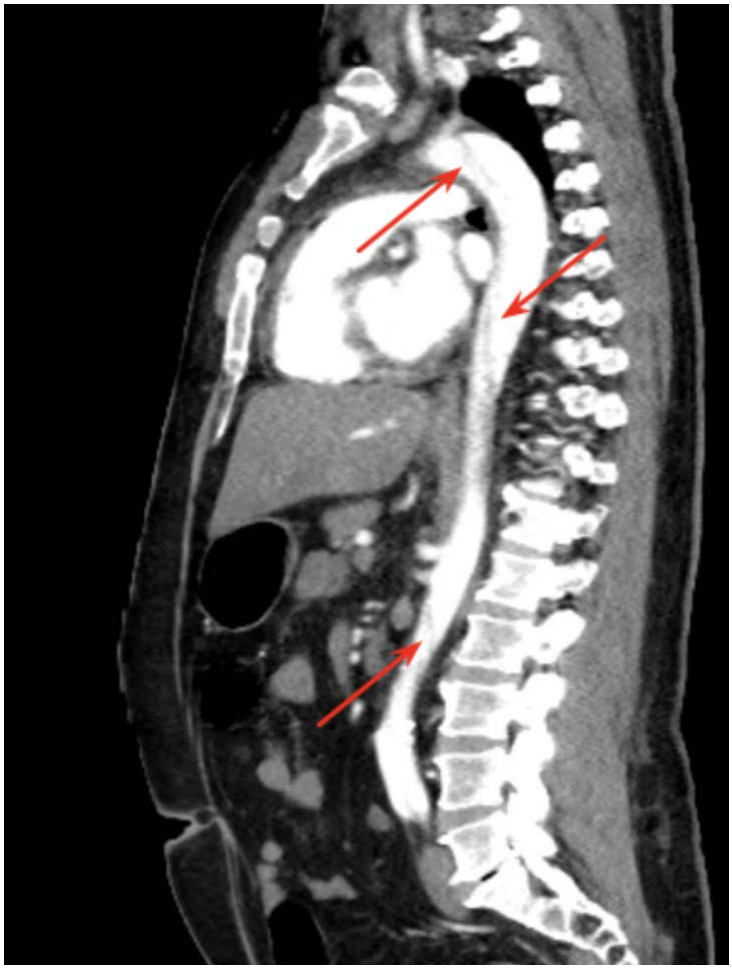

Figure 4. CTA showing a Type A aortic dissection extending along the entire length of the aorta

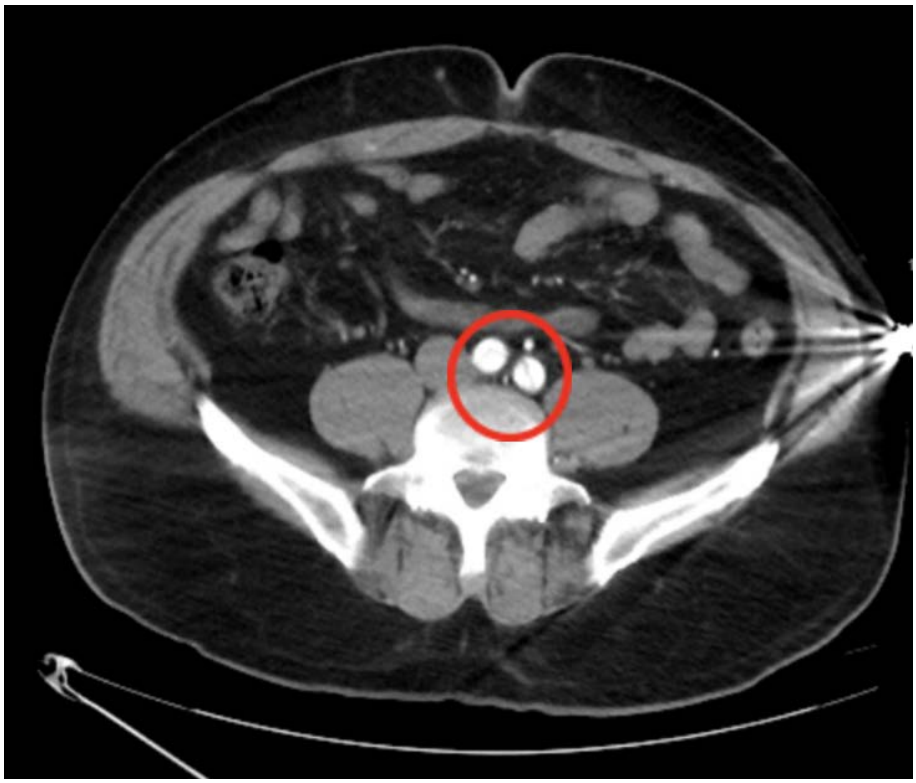

Figure 5. Extension of the dissection to iliac bifurcation

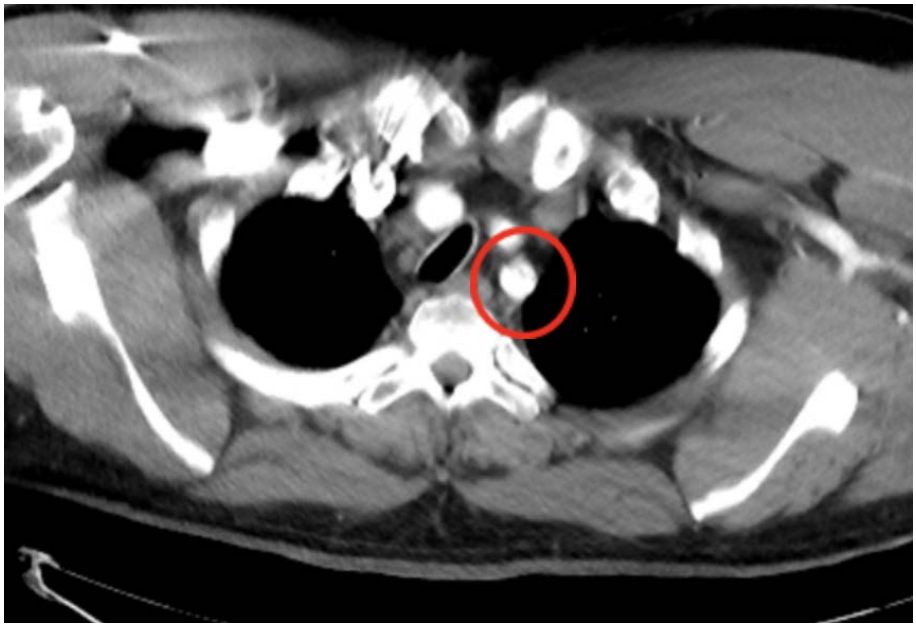

Figure 6. Extension of the dissection to the left subclavian artery

2. Discussion

AD occurs when there is a separation of the intimal layer from the rest of the aortic wall. Approximately 50-65% of ADs occur in the ascending aorta [1]. From the site of the initial tear, dissection can progress proximally to involve the aortic valve or distally to involve rest of the aorta and its branches [2]. Hypertension and male gender are the biggest risk factors associated with AD [3]. With the initial tear in the intimal layer, persistent hypertension can make the separation progress all along the aorta and its branches, creating a false lumen. False lumen can rapidly progress in size and compress the true lumen, causing ischemia in various organs supplied by the aorta [4]. Symptoms of AD depend on the extent of the dissection and the branches that it comprises. Sudden onset chest pain is the most common presenting symptom of AD, even though painless AD has also been reported in the literature [5]. Although rare, if AD occludes the opening of the coronaries, the initial presentation may be similar to an AMI with ischemic changes on EKG. STEMI as an initial presentation of AD is very rare and occurs in about 1-2% of cases [6]. Although both AD and AMI carry significant mortality if left untreated, distinction between the two entities prior to managing each is of crucial importance because treatment of AMI

with anticoagulation can be detrimental in a patient who actually has AD [7].

However, AD (especially type A) is one of the surgical emergencies that can be extremely difficult to diagnose. Diseases of the aorta have been referred to as "the great masquerader" due to their ability to present very similar to other common diseases of the chest and abdomen [8]. Making the correct diagnosis early is essential because the mortality rate increases 1-2% per hour within the first 24-48 hours after symptom onset [6]. CXR, which is often obtained in a patient complaining of chest pain, can reveal a widened mediastinum in up to 90% of cases [1]. Currently, the diagnosis of AD is made using CTA as it is readily available in the ED [9].

Our patient had a classic presentation of AMI with chest pain radiating to the jaw. EKG findings and laboratory data also seemed to support the diagnosis of AMI. Family history of a cardiac event in his father further strengthened our suspicion of AMI. Besides the mildly and untreated elevated blood pressure, there were no other clues in his presentation that could raise the possibility of AD. No widening of the mediastinum on his CXR further leads against the diagnosis of AD. In retrospect, failure of chest pain resolution despite being on a titratable nitroglycerin drip, should prompt the broadening of the differential diagnosis and to also include AD. Furthermore, in this case, jaw pain suggestive of classic AMI was presumably due to extension of the dissection to the left subclavian artery. We also suspect that his dissection may not have been as extensive as it was seen on imaging when he first arrived with chest pain to the ED. Delay in diagnosis and use of anticoagulation for a presumed AMI are potential causes of extension of AD. Fortunately, our patient had a good outcome from rapid surgical intervention when the appropriate diagnosis was made and was discharged home successfully. Younger age and lack of other comorbid conditions were likely contributing factors to his favorable outcome.

In our discussion, we would also like to review the use of point-of-care ultrasound (POCUS) and other methods to hasten the diagnosis of AD. *Chenkin* demonstrated the importance of POCUS in diagnosing AD in a patient who's presentation was that of a STEMI [10]. In their case, the original intention was to evaluate the left ventricular function of the heart in an ongoing STEMI, however, they found an echogenic flap in the ascending aorta, raising the suspicion for AD as confirmed with a CTA. Sensitivity and specificity of diagnosing ascending AD by transthoracic echocardiography are 78-90% and 87-96%, respectively [11]. Alternatively, if a patient with presumed AMI is triaged relatively early for cardiac catheterization, a quick check of the pressure difference between the aorta and the access site (either radial or femoral) can also reveal the diagnosis of AD. *Huang et.al.* demonstrated a significant pressure difference between the radial artery and aorta as a clue towards AD masquerading as STEMI [12]. They found the radial pressure to be 20 mmHg lower than the aortic pressure. This pressure difference is presumably due to much of the stroke volume going into the false lumen and not making its way to the peripheral arteries.

3. Conclusion

AD can be extremely difficult to diagnose due to its presentation being very similar to other common conditions of the chest and abdomen. Recognizing and managing AD early is of utmost importance due to the high mortality associated with AD. The clinical presentation of AD can resemble that of AMI, and differentiating between the two conditions is essential since management of AMI with anticoagulation can be lethal in a patient with AD. POCUS can be an effective method of diagnosing AD as it can be used to assess both the mechanical function of the heart and to evaluate for aortic pathology. Our case reiterates the importance of considering AD on the differential diagnosis, even in patients with a classic presentation of AMI.

Financial Source

No financial source was provided for this manuscript.

Disclosure

There are no actual or potential conflict of interest in relation to this manuscript.

Acknowledgments

Dr. Eli Ehrenpreis, MD

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